**PARAGRAPHS 19, 20, AND 21 OF OFFICE ACTION**

The Examiner objected to the specification under 35 U.S.C. §112, first paragraph, for reasons of record. Additionally, the Examiner rejected claims 1, 3, 5-10, 15, 17-19, 23-24, 28-32, 35, 37-42 and 78 under 35 U.S.C. § 112, first paragraph, for the reasons set forth in the objection to the specification.

**APPLICANTS HAVE MET THE LEGAL STANDARD**

**SUBPARAGRAPH A**

**USE OF MONOCLONAL ANTIBODIES IN THE CLAIMED METHODS**

Applicants respectfully disagree with the Examiner's position concerning the inoperability of using monoclonal antibodies in the claimed methods. However, in order to further the prosecution of the subject application, applicants amended the claims to exclude the use of monoclonal antibodies. Accordingly, the rejection as to this matter is rendered moot.

**USE OF B7 AND CD28 ANTIGENS IN THE CLAIMED METHODS**

The Examiner states that there is a high degree of unpredictability associated with the use of proteins in vivo for the following reasons:

> "...(1) the protein may be inactivated before producing an effect, i.e. such as proteolytic degradation, immunological inactivation or due to an inherently short half life of the protein; (2) the protein may otherwise not reach the target area because, for example, (a) the protein may not be able to cross the mucosa, (b) the protein may be adsorbed or absorbed by fluids, cells and tissues where the protein has no effect; and (3) other functional properties, known or unknown, may make the protein unsuitable for <u>in vivo</u> use" (Office Action

at page 3, lines 20-27).

In particular, the use of B7Ig and CD28Ig fusion proteins in vivo allegedly involves a high degree of unpredictability for the following reasons:

> "...(1) the protein may be inactivated before producing an effect, i.e. such as proteolytic degradation, immunological inactivation or due to an inherently short half life of the protein; (2) the protein may otherwise not reach the target area because, for example, (a) the protein may not be able to cross the mucosa, (b) the protein may be adsorbed or absorbed by fluids, cells and tissues where the protein has no effect; and (3) other functional properties, known or unknown, may make the protein unsuitable for in vivo use" (Office Action at page 4, lines 25-36).

Before addressing the Examiner's basis for rejection on the merits, applicants respectfully contend that the basis for rejecting the claims is improper. Although the Examiner withdrew the rejection under 35 U.S.C. §101, the basis for the rejection under 35 U.S.C. §112, first paragraph, is deeply rooted in terms of utility and operability under 35 U.S.C. §101.

Withdrawing the §101 rejection but maintaining the §112 rejection using arguments rooted in utility and operability is improper and the rejection under section 112, first paragraph should be withdrawn together with the correlative rejection under section 101[1].

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[1] Final PTO Utility Examination Guidelines (60 FR 36263).

It is well established that an invention that is inoperative (i.e., it does not operate to produce the results claimed by the patent applicant) is not a useful invention in the meaning of patent law[2]. However, as the Federal Circuit stated "[t]o violate §101 the claimed device must be totally incapable of achieving a useful result."[3] In this case, there is no basis to believe that the claimed methods are totally incapable of achieving a useful result. In fact, applicants' in vitro data shows otherwise.

Cases decided by a Federal court in which a claimed invention was held to lack utility under §101 because it was "inoperative" have been rare. Uniformly, in these cases the utility asserted by the applicant was "incredible in the light of knowledge of the art, or factually misleading"[4] when initially considered by the Examiner. Examples include: an invention asserted to change the taste of

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[2] Newman v. Quigg, 877 F.2d 1575, 1581, 11 USPQ2d 1340, 1345 (Fed. Cir. 1989).

[3] Brooktree Corp. v. Advanced Micro Devices, Inc., 977 F.2d 1555, 24 USPQ2d 1401, 1412 (Fed Cir. 1992) (emphasis added). See also, E.I. du Pont De Nemours and Co. v. Berkley and Co., 620 F.2d 1247, 1260 n.17, 205 USPQ 1, 10 n.17 (8th Cir. 1980) ("A small degree of utility is sufficient. The claimed invention must only be capable of performing some beneficial function . . . An invention does not lack utility merely because the particular embodiment disclosed in the patent lacks perfection or performs crudely . . . A commercially successful product is not required . . . Nor is it essential that the invention accomplish all its intended functions . . . or operate under all conditions . . . partial success being sufficient to demonstrate patentable utility . . . In short, the defense of non-utility cannot be sustained without proof of total incapacity" [citations omitted]).

[4] In re Citron, 325 F.2d 248, 253, 139 USPQ 516, 520 (CCPA 1963).

food using a magnetic field,[5] a perpetual motion machine,[6] a method for increasing the energy output of fossil fuels upon combustion through exposure to a magnetic field,[7] uncharacterized compositions for curing cancer[8] and a method of restoring hair growth.[9] In view of the rare nature of such cases, Examiners should not label an asserted utility "incredible" unless it is clearly appropriate to do so.

In this case, there is no basis for the Examiner to assert that the claimed methods constitute an incredible use. The possible problems with using proteins in vivo as stated by the Examiner (e.g., possible inactivation of the protein, questions of half life, ability of protein to reach its target, the protein's lack of ability to cross mucosa) are problems generally associated with the use of all compositions. These potential problems are not particular to the use of proteins. In view of this broad generalization, the basis for the rejection is not specific to the claimed invention and is improper and should be withdrawn. The Examiner has not questioned whether applicants have taught how to use the claimed methods but only whether the proteins used in the claimed methods will work.

Applicants respectfully contend that the basis for the rejection under section 112, first paragraph, is an issue of operability and

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[5] Fregeau v. Mossinghoff, 776 F.2d 1034, 227 USPQ 848 (Fed. Cir. 1985).

[6] Newman v. Quigg, 877 F.2d at 1581, 11 USPQ2d at 1340.

[7] In re Ruskin, 354 F.2d 395, 148 USPQ 221 (CCPA 1966).

[8] In re Citron, 325 F.2d 248, 139 USPQ 516 (CCPA 1963).

[9] In re Ferens, 417 F.2d 1072, 163 USPQ 609 (CCPA 1969).

and in view of the rare nature of such cases due to the stringent standards for asserting inoperability, applicants respectfully contend that the rejection is improper and should be withdrawn.

Moreover, even if it were proper to maintain the rejection under section 112, first paragraph, despite the withdrawal of the corresponding rejection under section 101, the rejection under section 112, first paragraph, fails because, without more, the basis for the rejection is merely an unsupported assertion. The Patent Office recites various difficulties which might be encountered in attempting to put the invention into practice and further asserts that there might be still other difficulties which could not have been foreseen.

These statements do not constitute a sufficiently definite statement of a basis for rejection[10].

The case law is clear, the Patent Office must treat as true applicants' asserted use, unless:

1. the mode of operation of the claimed invention cannot be readily understood and does not conform to the known laws of physics and chemistry,
2. the mode of operation of the claimed invention operation conflicts with a recognized scientific principle as for example where an applicant purports to have discovered a machine producing perpetual motion, the presumption of inoperativeness is so strong that very clear evidence is required to overcome it,
3. the invention is of such a nature that it could not be

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[10] In re Chilowsky, 108 USPQ 321, 325 (1956).

tested by any known scientific principles[11].

In this regard, applicants respectfully point out that paragraph 1 above has not been met because the use of proteins, in general, and Ig fusion proteins, in particular, are well known and accepted (applicants' response dated August 28, 1995, at pages 11-12).

Further, paragraph 2 above has not been met because the claimed methods are not limited to in vivo uses and they do not conflict with a recognized scientific principle. In fact, applicants' in vitro data confirm the operability of the claimed methods (specification at page 64, lines 21-25, lines 27-30; page 71, lines 5-9, lines 28-29, lines 29-35). Further, applicants provided in vivo data confirming the in vitro results using a homologous molecule, namely, CTLA4Ig (see applicants' response dated August 24, 1992 of parent application, namely, U.S. Serial No. 722,101). CTLA4 is homologous to CD28. This in vivo data strengthens applicants' in vitro data.

There is no reason to believe that the use of proteins, in general, or B7 and CD28 antigens, in particular, would be unpredictable in view of the successful use of soluble homologous molecules, e.g., CTLA4, in vivo. In accordance with Brana, applicants' in vitro evidence alone should be sufficient to satisfy applicants' burden[12]. In combination with in vivo data concerning homologous molecules, there is no reason to doubt applicants' assertion.

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[11] In re Chilowsky, supra; In re Brana et al., 34 USPQ2d 1438 (CAFC 1995).

[12] In re Brana at page 1442.

**SUBPARAGRAPH B**

The Examiner has taken the position that the specification does not support a method of inhibiting T cell proliferation with any B7 antigen derivative. The Examiner suggested limiting the claimed invention to the use of B7Ig.

Applicants traverse the basis for the objection to the specification and rejection of the pending claims for the reasons which follows.

Applicants' invention involves the discovery that blocking the CD28/CTLA4/B7 pathways results in the inhibition of T cell proliferation. In accordance with this discovery, the claims recite the use of both B7 and CD28 antigens.

In the case of using B7 antigens, applicants are justly entitled to more than the use of B7Ig. Clearly, the invention is directed to the discovery that B7 will recognize CD28 and that this recognition produces the claimed result.

B7Ig is but one embodiment of soluble B7 molecules (which bind to CD28). Once those skilled in the art knew applicants' discovery, it would have been well within their skill to make other soluble B7 molecules which bind to CD28 to effect the same result as that which applicants' achieved using B7Ig because methods for making soluble proteins were known[13].

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[13] Capon et al., "Designing CD4 Immunoadhesins for AIDS Therapy" Nature, 337:525 (1989) already of record, cited in the Office Action dated December 12, 1992 in parent application U.S. Serial No. 07/722,101); Capon et al. WO 89/02922 already of record, cited in the Office Action dated April 26, 1995).

Applicants respectfully contend that applicants need not teach what is well known in the art[14]. Methods of making recombinant proteins, e.g., soluble proteins, were well known to those skilled in the art well before applicants' invention.

In fact applicants teach that:

> "The B7 antigen and/or its fragments or derivatives for use in the present invention may be produced in recombinant form using known molecular biology techniques based on the cDNA sequence published by Freeman et al., supra. Specifically, cDNA sequences encoding the amino acid sequence corresponding to the B7 antigen or fragments or derivatives thereof can be synthesized by the polymerase chain reaction (see U.S. Patent No. 4,683,202) using primers derived from the published sequence of the antigen (Freeman et al., supra). These cDNA sequences can then be assembled into a eukaryotic or prokaryotic expression vector and the resulting vector can be used to direct the synthesis of the ligand for CD28 by appropriate host cells, for example COS or CHO cells. CD28 receptor and/or its fragments or derivatives may also be produced using recombinant methods" (specification at page 12, lines 20-34).

**THE INVENTION LIES IN THE DISCOVERY THAT THE COUNTER RECEPTOR FOR THE B7 MOLECULE IS CD28**

Using applicants' discovery that the counter receptor for the B7 molecule is CD28, it would have been routine to make and use recombinant B7 proteins capable of binding CD28 and recombinant CD28 proteins capable of binding B7. Identifying recombinant soluble B7 proteins which bind to CD28 merely involves setting up competition assays in which the B7Ig of the present invention is used to compete with other embodiments of soluble B7 proteins for CD28.

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[14] Staehelin v. Secher, 24 U.S.P.Q.2d 1513, 1516 (Bd. Pat. App. & Int. 1992).

Competition assays are routinely done and their protocols are clearly within the skill of one in the art (E. Harlow and D. Lane, eds., "Antibodies: A Laboratory Manual" 1988, pages 567-577 already of record as Exhibit 2 of applicants' December 13, 1993 response).

Any recombinant B7 protein having at least a portion of the B7 antigen which recognizes and binds CD28 fall within the scope of this invention (specification at page 23, lines 22-25).

In fact, the general technique for construction of expression vectors for fusion proteins were well known in the art (Ernst Winnacker, "From Genes to Clones: Introduction to Gene Technology" Chapter 7, (1987) at pages 279-305 annexed herewith as Exhibit 1).

The general approach for the construction of expression vectors directing the synthesis of fusion proteins is as follows (Ernst-L. Winnacker, supra at page 291).

The starting material can be a B7 cDNA clone whose B7 insertion has been removed from a vector (Ernst-L. Winnacker, supra at page 291). Using methods known in the art a restriction site is positioned close to a start codon. The next step is a digestion step which should cut DNA fragments asymmetrically. The mixture of DNA fragments obtained is then cloned into a vector, e.g., a pUC vector. Of course, a cleavage site must be present within the polylinker of the chosen vector. Since a wide spectrum of vectors is available, it should not be difficult to find a suitable vector containing the desired cleavage site. Once a suitable clone is identified, the cleavage site can be used for the insertion of the gene of interest, which can be obtained from the original cDNA clone.

Applicants respectfully contend that the DNA and amino acid sequence for B7 was published before the filing date of the subject application. Therefore, because of applicants' discovery, it would have been routine to generate soluble B7 proteins for the claimed methods using standard techniques (e.g., see Sambrook et al., eds., Molecular Cloning, A Laboratory Manual, 2nd Edition, Cold Spring Harbor Laboratory Press (1989)).

In fact, as a guideline applicants teach exemplary and routine methods to generate soluble B7 and CD28 proteins as follows:

> "The techniques for assembling and expressing DNA encoding the amino acid sequences corresponding to B7 antigen and soluble B7Ig and CD28Ig fusion proteins, e.g synthesis of oligonucleotides, PCR, transforming cells, constructing vectors, expression systems, and the like are well-established in the art, and most practitioners are familiar with the standard resource materials for specific conditions and procedures. However, the following paragraphs are provided for convenience and notation of modifications where necessary, and may serve as a guideline.
>
> Cloning and Expression of Coding Sequences for Receptors and Fusion Proteins
>
> cDNA clones containing DNA encoding CD28 and B7 proteins are obtained to provide DNA for assembling CD28 and B7 fusion proteins as described by Aruffo and Seed, Proc. Natl. Acad. Sci. USA 84:8573-8579 (1987) (for CD28); and Freeman et al., J. Immunol. 143:2714-2722 (1989) (for B7), incorporated by reference herein. Alternatively, cDNA clones may be prepared from RNA obtained from cells expressing B7 antigen and CD28 receptor based on knowledge of the published sequences for these proteins (Aruffo and Seed, and Freeman, supra) using standard procedures.
>
> The cDNA is amplified using the polymerase chain reaction ("PCR") technique (see U.S. Patent Nos. 4,683,195 and 4,683,202 to Mullis et al. and Mullis & Faloona, Methods Enzymol. 154:335-350 (1987)) using synthetic oligonucleotides encoding the sequences corresponding to the extracellular domain of the CD28 and B7 proteins as primers. PCR is then used to adapt the fragments for ligation to the DNA encoding amino acid fragments corresponding to the human

immunoglobulin constant γ 1 region, i.e. sequences encoding the hinge, CH2 and CH3 regions of Ig Cγ1 to form B7Ig and CD28Ig fusion constructs and to expression plasmid DNA to form cloning and expression plasmids containing sequences corresponding to B7 or CD28 fusion proteins.

To produce large quantities of cloned DNA, vectors containing DNA encoding the amino acid sequences corresponding to the fusion constructs of the invention are transformed into suitable host cells, such as the bacterial cell line MC1061/p3 using standard procedures, and colonies are screened for the appropriate plasmids.

The clones obtained as described above are then transfected into suitable host cells for expression. Depending on the host cell used, transfection is performed using standard techniques appropriate to such cells. For example, transfection into mammalian cells is accomplished using DEAE-dextran mediated transfection, $CaPO_4$ co-precipitation, lipofection, electroporation, or protoplast fusion, and other methods known in the art including: lysozyme fusion or erythrocyte fusion, scraping, direct uptake, osmotic or sucrose shock, direct microinjection, indirect microinjection such as via erythrocyte-mediated techniques, and/or by subjecting host cells to electric currents. The above list of transfection techniques is not considered to be exhaustive, as other procedures for introducing genetic information into cells will no doubt be developed.

Expression plasmids containing cDNAs encoding sequences corresponding to CD28 and B7 for cloning and expression of CD28Ig and B7Ig fusion proteins include the OMCD28 and OMB7 vectors modified from vectors described by Aruffo and Seed, Proc. Natl. Acad. Sci. USA (1987), supra, (CD28); and Freeman et al., (1989), supra, (B7), both of which are incorporated by reference herein. Preferred host cells for expression of CD28Ig and B7Ig proteins include COS and CHO cells.

Expression in eukaryotic host cell cultures derived from multicellular organisms is preferred (see Tissue Cultures, Academic Press, Cruz and Patterson, Eds. (1973)). These systems have the additional advantage of the ability to splice out introns and thus can be used directly to express genomic fragments. Useful host cell lines include Chinese hamster ovary (CHO), monkey kidney (COS), VERO and HeLa cells. In the present invention, cell lines stably expressing the fusion constructs are preferred.

Expression vectors for such cells ordinarily include promoters and control sequences compatible with mammalian cells such as, for example, CMV promoter (CDM8 vector) and avian sarcoma virus (ASV) (πLN vector). Other commonly used early and late promoters include those from Simian Virus 40 (SV 40) (Fiers, et al., Nature 273:113 (1973)), or other viral promoters such as those derived from polyoma, Adenovirus 2, and bovine papilloma virus. The controllable promoter, hMTII (Karin, et al., Nature 299:797-802 (1982)) may also be used. General aspects of mammalian cell host system transformations have been described by Axel (U.S. Patent No. 4,399,216 issued Aug. 16, 1983). It now appears, that "enhancer" regions are important in optimizing expression; these are, generally, sequences found upstream or downstream of the promoter region in non-coding DNA regions. Origins of replication may be obtained, if needed, from viral sources. However, integration into the chromosome is a common mechanism for DNA replication in eukaryotes.

Although preferred host cells for expression of the DNA constructs include eukaryotic cells such as COS or CHO cells, other eukaryotic microbes may be used as hosts. Laboratory strains of Saccharomyces cerevisiae, Baker's yeast, are most used although other strains such as Schizosaccharomyces pombe may be used. Vectors employing, for example, the 2μ origin of replication of Broach, Meth. Enz. 101:307 (1983), or other yeast compatible origins of replications (see, for example, Stinchcomb et al., Nature 282:39 (1979)); Tschempe et al., Gene 10:157 (1980); and Clarke et al., Meth. Enz. 101:300 (1983)) may be used. Control sequences for yeast vectors include promoters for the synthesis of glycolytic enzymes (Hess et al., J. Adv. Enzyme Reg. 7:149 (1968); Holland et al., Biochemistry 17:4900 (1978)). Additional promoters known in the art include the CMV promoter provided in the CDM8 vector (Toyama and Okayama, FEBS 268:217-221 (1990); the promoter for 3-phosphoglycerate kinase (Hitzeman et al., J. Biol. Chem. 255:2073 (1980)), and those for other glycolytic enzymes. Other promoters, which have the additional advantage of transcription controlled by growth conditions are the promoter regions for alcohol dehydrogenase 2, isocytochrome C, acid phosphatase, degradative enzymes associated with nitrogen metabolism, and enzymes responsible for maltose and galactose utilization. It is also believed terminator sequences are desirable at the 3' end of the coding sequences. Such terminators are found in the 3' untranslated region following the coding sequences in yeast-derived genes.

Alternatively, prokaryotic cells may be used as hosts for expression. Prokaryotes most frequently are represented by various strains of E. coli; however, other microbial strains may also be used. Commonly used prokaryotic control sequences which are defined herein to include promoters for transcription initiation, optionally with an operator, along with ribosome binding site sequences, include such commonly used promoters as the beta-lactamase (penicillinase) and lactose (lac) promoter systems (Chang et al., Nature 198: 1056 (1977)), the tryptophan (trp) promoter system (Goeddel et al., Nucleic Acids Res. 8:4057 (1980)) and the lambda derived $P_L$ promoter and N-gene ribosome binding site (Shimatake et al., Nature 292:128 (1981)).

The nucleotide sequences encoding the amino acid sequences corresponding to the CD28Ig and B7Ig fusion proteins, may be expressed in a variety of systems as set forth below. The cDNA may be excised by suitable restriction enzymes and ligated into suitable prokaryotic or eukaryotic expression vectors for such expression. Because CD28 receptors occur in nature as dimers, it is believed that successful expression of these proteins requires an expression system which permits these proteins to form as dimers. Truncated versions of these proteins (i.e. formed by introduction of a stop codon into the sequence at a position upstream of the transmembrane region of the protein) appear not to be expressed. The expression of CD28 antigen in the form of a fusion protein permits dimer formation of the protein. Thus, expression of CD28 antigen as a fusion product is preferred in the present invention.

Sequences of the resulting fusion protein constructs are confirmed by DNA sequencing using known procedures, for example as described by Sanger et al., Proc. Natl. Acad. Sci. USA 74:5463 (1977) as further described by Messing et al., Nucleic Acids Res. 9:309 (1981) or by the method of Maxam et al. Methods Enzymol. 65:499 (1980)).

Recovery of Protein Products

As noted above, the CD28 receptor is not readily expressed as a mature protein using direct expression of DNA encoding the amino acid sequence corresponding to the truncated protein. To enable homodimer formation, it is preferred that DNA encoding the amino acid sequence corresponding to the extracellular domain of CD28 and including the codons for a signal sequence such as oncostatin M in cells capable of appropriate processing, is fused with DNA encoding amino

acids corresponding to the Fc domain of a naturally dimeric protein. Purification of the fusion protein products after secretion from the cells is thus facilitated using antibodies reactive with the anti-immunoglobulin portion of the fusion proteins. When secreted into the medium, the fusion protein product is recovered using standard protein purification techniques, for example by application to protein A columns)" (specification at pages 18-20)

**SUBPARAGRAPH I**

Contrary to the Examiner's position, the presence of endogenous CTLA4 does not destroy the operability of the claimed method.

B7 binds both CD28 and CTLA4. Therefore, the Examiner's comments concerning whether the CTLA4/B7 interaction can activate T cells is assuaged as to claims directed to using a B7 antigen, namely claims 1, 3, 5-8, 41, and 42. Clearly, a soluble B7 molecule would bind CD28 and CTLA4.

With regard to claims directed to using a CD28 antigen, namely, claims 19, 40, and 78, the rejection is misplaced. It is true that a soluble CD28 molecule would bind B7-positive cells thereby forming a CD28/B7-positive cell complex resulting in inhibition of T cell proliferation. It is also true that a soluble CTLA4 molecule would bind B7-positive cells thereby forming a CD28/B7-positive cell complex resulting in inhibition of T cell proliferation (Linsley et al., J. Exp. Med. 174:561-659 (1991)). Therefore, in either scenario, T cell proliferation is inhibited as claimed.

Keep in mind that amended claims 19 (including dependent claim 40) and 78 require reacting B7 positive cells with exogenous CD28 antigen. This means exogenous CD28 antigen is being added into the system. There is no requirement in the claims for a similar

increase in CTLA4. Therefore, addition of exogenous CD28 antigen pushes the equilibrium toward exogenous CD28 binding B7.

The Examiner's observation that the claimed methods are unpredictable because CTLA4/B7 interaction can still activate T cells is misplaced and should be withdrawn.

**PRIOR ART REJECTIONS**

Applicants are pleased that the Examiner has withdrawn the rejection of claims 1, 3, 5-8, 18, 23, 24, 41 and 42 under 35 U.S.C. § 103 as allegedly unpatentable over Linsley et al. [PNAS 87:5031-5035 (1990)] and Freeman et al. [J. Immunology 143:2714-1722 (1989)] in view of Capon et al. [WO 89/02922].

Applicants are pleased that the Examiner has withdrawn the rejection of claims 19 and 78 under 35 U.S.C. § 103 as allegedly unpatentable over Linsley et al. [PNAS 87:5031-5035 (1990)] and Aruffo et al. [PNAS 84:8573-8577 (1987)] in view of Capon et al. [WO 89/02922].

The Examiner maintained the rejection of claims 35 and 37-40 under 35 U.S.C. § 102(b) as allegedly anticipated by Damle et al. [J.I. 140(6):1753-1761 (1988)].

Applicants respectfully disagree with the Examiner's position. However, in order to further the prosecution of the subject application, applicants amended claims 35 and 37-40 excluding the use of monoclonal antibodies from these claims. Accordingly, the rejection is rendered moot.

The Examiner maintained the rejection of claims 1 and 37-40 under

35 U.S.C. § 102(b) as allegedly anticipated by Ledbetter et al. [J.I. 135(4):2331-2335 (1985)] for reasons of record.

Applicants respectfully disagree with the Examiner's position. However, in order to further the prosecution of the subject application, applicants amended claims 1 and 37-40 by excluding the use of monoclonal antibodies therein. Accordingly, the rejection is rendered moot.

No fee, other than the extension fee, is deemed necessary in connection with the filing of this response. If any fee is necessary, the Patent Office is authorized to charge any additional fee to Deposit Account No. 13-2724.

Respectfully submitted,

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